Changes in the human muscle force-velocity relationship in response to resistance training and subsequent detraining

Lars L. Andersen,1,2 Jesper L. Andersen,2 S. Peter Magnusson,1 Charlotte Suettà,1 Jørgen L. Madsen,2 Lasse R. Christensen,2 and Per Aagaard1

1Institute of Sports Medicine Copenhagen/Team Danmark Testcenter, Bispebjerg Hospital, and 2Copenhagen Muscle Research Center, Rigshospitalet, Copenhagen, Denmark

Submitted 26 January 2005; accepted in final form 22 February 2005

PREVIOUS STUDIES HAVE INVESTIGATED various physiological effects of detraining subsequent to resistance training. Reductions in maximal muscle strength (20, 31, 35, 49), muscular peak power production (41, 47), muscle size (8, 34, 49), and neural drive to the muscle (29, 31, 49) have been reported. Consequently, a faster muscle contractile behavior may be expected with detraining subsequent to resistance training. However, during maximal unloaded movements, the functional benefit of this fiber-type transformation may be overruled by muscular atrophy, decreased muscle strength, and decreased neural drive. The effect of detraining on maximal unloaded limb movement speed and power has not been examined previously. It can be hypothesized that motor tasks involving maximal movement speed and negligible external resistance (e.g., kicking, punching, smashing) would be enhanced with detraining in consequence of a faster muscle fiber-type composition.

The aim of the present study was to examine the effect of detraining subsequent to resistance training on slow-to-medium-velocity isokinetic muscle strength vs. performance of maximal-velocity unloaded limb movement. Muscle cross-sectional area (CSA), agonist and antagonist electromyographic (EMG) activity, evoked muscle twitch characteristics, and MHC isoform composition were obtained to elucidate the mechanism of the underlying adaptation.

METHODS

Subjects

Fourteen healthy sedentary male subjects (age 23.6 ± 3.1 yr, stature 179 ± 8 cm, body mass 75.8 ± 8.5 kg, means ± SD) participated in the training study. Furthermore, 10 subjects (age 23.9 ± 3.1 yr, stature 181 ± 8 cm, body mass 76.0 ± 5.0 kg) served as control group. None of the subjects had previously participated in regular resistance training, and none of the subjects had participated in regular exercise within the last year before entering the study. Most subjects used a bicycle for daily transportation (10–20 min per day). All subjects gave written, informed consent to participate in the study, which was approved by the local Ethics Committee (KF-01-010/01).

Resistance Training

Resistance training was performed for 3 mo with 38 sessions evenly dispersed, followed by 3 mo of detraining (= no training). The training program consisted of four to five sets of incline leg press,
hack squat, isolated knee extension, and hamstring curl. All exercises were performed in a traditional manner using cyclic concentric and eccentric muscle contractions, i.e., raising and lowering the weight stack in a controlled manner without pause or breaks. In the early training phase (sessions 1–15) exercises consisted of 10–12 repetition maximum (RM) loads (4 sets), followed by 8–10 RM loads (4 sets) in the midphase (sessions 16–25), and heavier loads of 6–8 RM (5 sets) in the later phase (sessions 26–38) (8). Absolute training loads were progressively increased to maintain relative loadings at the intended level. To ensure adequate training load and intensity, all training sessions were surveyed and supervised by the authors of the study.

**Detraining from Resistance Training**

The period of resistance training was followed by 3 mo of detraining. The subjects were instructed to return to their usual lifestyle and level of physical activity during this period.

**Muscle Strength Testing**

Isokinetic dynamometry was used to evaluate concentric moment of force at angular velocities of 30 and 240°/s for the knee extensors of the right leg (Kinetics Communicator, Chatricec, Chattanooga, TN) (5). Range of motion was 10 to 90° anatomical knee joint angle (0° = full extension). Successive maximal trials were performed, separated by rest periods of at least 45 s until peak moment could not be improved any further, which was usually achieved within 6–8 maximal attempts. Dynamometer strain gauge and lever arm position signals were sampled synchronously at 1,000 Hz using an external analog-to-digital-converter (DT9804, Data Translation, Marlboro, MA). During offline analysis, the dynamometer force and lever arm position signal were digitally low-pass filtered with a 15- and 8-Hz cutoff frequency, respectively. Subsequently, the moment of force signal was corrected for the effect of gravity (7). The average moment exerted in the 60–80° interval was calculated. The rationale for choosing this knee joint angle interval was that the highest moment of force occurred in this particular range of motion.

**Maximal Unloaded Knee Extension**

Angular velocity, acceleration, moment of force, and power development during maximal unloaded knee extension were determined on the basis of goniometry and classical biomechanical principles. A flexible goniometer (Penny & Giles, Christchurch, Dorset, UK) was positioned laterally at the right knee joint. Subjects were seated in a rigid chair with the backrest 10° reclined. The leg was strapped at the distal part of the thigh and hip to exclude movements of the hip joint. The lower leg was allowed to move freely. Subjects were instructed to extend their right knee “as fast as possible” into a firm foam pillow. The movement range was from 90°, which was the relaxed starting position, to ~5–10° from full extension. Ten maximal unloaded knee extensions were performed separated by rest periods of 30 s. Trials with a visible countermovement were discarded. Goniometer position signals were sampled at 1,000 Hz (DT9804, Data Translation). During offline analysis, the goniometer position signal was digitally low-pass filtered at a cutoff frequency of 10 Hz using a fourth-order zero phase lag Butterworth filter (62). The goniometer signal was mathematically integrated (iEMG, unit μV·s) in the 60–80° knee joint angle. To compare between contraction speeds, the iEMG was divided by integration time to yield average EMG in the 60–80° interval. Identical changes in VL, VM, and RF EMG were observed with training, and therefore EMG data were collapsed, i.e., quadriceps EMG = (VL + VM + RF EMG)/3.

**Normal EMG.** To compare EMG signal amplitudes between contractions at slow, medium, and maximal velocity, EMG were normalized relative to the EMG amplitude measured during maximal concentric contraction at 240°/s (5, 53).

**Antagonist EMG.** To quantify the degree of antagonist muscle involvement, EMG signals were synchronously obtained in the biceps femoris caput longus and semitendinosus (5).

**Muscle CSA**

Quadriiceps anatomical CSA was determined at midfemur (3, 48) from two-dimensional T1-weighted fast field echo (repetition time/echo time, 500/14 ms; field of view 180; matrix 512 × 512; slice thickness 6 mm) magnetic resonance images (MRI) (Philips, Gyroscan ACS-NT 1.5 T, Best, Holland) at pre- and posttraining and post-detraining. During analysis, CSAs of individual muscles of the thigh were determined by using the computer software of the MRI scanner. CSA of the quadriceps muscle was defined as the sum of CSA of the VL, RF, VM, and vastus intermedius muscles. Financial circumstances precluded obtaining MRI data for the control group.

**Biopsy Sampling and MHC Analysis**

Muscle biopsies were obtained from the middle portion of the right VL muscle (15) and immediately mounted with Tissue-Tek, frozen in isopentane cooled with liquid nitrogen, and stored at −80°C until processed. Pre- and posttraining and post-detraining biopsies were taken from the same region and depth of the VL muscle. MHC analysis was performed on the biopsies using SDS-PAGE. From each biopsy, 10–20 serial cross sections (20 μm) were cut in a cryostat and placed in 100–200 μl of lysing buffer and heated for 3 min at 90°C (27). Five to 20 μl of the myosin-containing samples were loaded on a SDS-PAGE gel containing 6% polyacrylamide and 30% glycerol. Gels were run at 70 V for 42 h at 4°C. MHC isoform content was determined with a densiometric system (Cream 1D, KemEnTec Aps, Copenhagen, Denmark) (8). These measurements were not performed for the nontrained control group.

**Evoked Muscle Twitch Measurements**

Electrically evoked twitch contractile properties of the VL muscle were determined. The subjects were seated in an upright position with expressed as average values during the 60–80° range of motion to permit comparison with the isokinetic test results. Furthermore, peak velocity was determined as the greatest slope over successive 2 ms of the goniometer position curve.

**Electromyography**

EMG signals were obtained during the isokinetic test and during the maximal unloaded knee extension. After careful preparation of the skin, bipolar surface EMG electrodes (Medicotest M-00-S) were placed on the medial portion of the vastus lateralis (VL), vastus medialis (VM), and rectus femoris (RF) muscles. The exact electrode position was registered and used during all test bouts. The EMG electrodes were connected directly to small preamplifiers located ~10 cm from the recording site. The signals were lead through shielded wires to custom-built differential instrumentation amplifiers, with a bandwidth of 10–10,000 Hz and a common mode rejection ratio >100 dB, and sampled at 1,000 Hz (5). During the process of offline analysis, all raw EMG signals were digitally high-pass filtered at a cutoff frequency of 5 Hz using a fourth-order zero phase lag Butterworth filter (62). The EMG signal was then passed through a symmetric moving root mean square filter with a time constant of 20 ms. The filtered EMG signal was mathematically integrated (iEMG, unit μV·s) in the 60–80° knee joint angle. To compare between contraction speeds, the iEMG was divided by integration time to yield average EMG in the 60–80° interval. Identical changes in VL, VM, and RF EMG were observed with training, and therefore EMG data were collapsed, i.e., quadriceps EMG = (VL + VM + RF EMG)/3.

**Normal EMG.** To compare EMG signal amplitudes between contractions at slow, medium, and maximal velocity, EMG were normalized relative to the EMG amplitude measured during maximal concentric contraction at 240°/s (5, 53).

**Antagonist EMG.** To quantify the degree of antagonist muscle involvement, EMG signals were synchronously obtained in the biceps femoris caput longus and semitendinosus (5).

**Muscle CSA**

Quadriiceps anatomical CSA was determined at midfemur (3, 48) from two-dimensional T1-weighted fast field echo (repetition time/echo time, 500/14 ms; field of view 180; matrix 512 × 512; slice thickness 6 mm) magnetic resonance images (MRI) (Philips, Gyroscan ACS-NT 1.5 T, Best, Holland) at pre- and posttraining and post-detraining. During analysis, CSAs of individual muscles of the thigh were determined by using the computer software of the MRI scanner. CSA of the quadriceps muscle was defined as the sum of CSA of the VL, RF, VM, and vastus intermedius muscles. Financial circumstances precluded obtaining MRI data for the control group.

**Biopsy Sampling and MHC Analysis**

Muscle biopsies were obtained from the middle portion of the right VL muscle (15) and immediately mounted with Tissue-Tek, frozen in isopentane cooled with liquid nitrogen, and stored at −80°C until processed. Pre- and posttraining and post-detraining biopsies were taken from the same region and depth of the VL muscle. MHC analysis was performed on the biopsies using SDS-PAGE. From each biopsy, 10–20 serial cross sections (20 μm) were cut in a cryostat and placed in 100–200 μl of lysing buffer and heated for 3 min at 90°C (27). Five to 20 μl of the myosin-containing samples were loaded on a SDS-PAGE gel containing 6% polyacrylamide and 30% glycerol. Gels were run at 70 V for 42 h at 4°C. MHC isoform content was determined with a densiometric system (Cream 1D, KemEnTec Aps, Copenhagen, Denmark) (8). These measurements were not performed for the nontrained control group.

**Evoked Muscle Twitch Measurements**

Electrically evoked twitch contractile properties of the VL muscle were determined. The subjects were seated in an upright position with...
the knee flexed 90°. A steel cuff was strapped around the lower leg, ~3 cm above the malleoli, and connected via a rigid steel bar to a strain gauge load cell (Bofors KRG-4, Bofors), which was connected to a preamplifier (BK15, Nobel Elektronik) and an amplifier (Gould 5900, Gould, Valley View, OH). Surface stimulation electrodes (Bioflex, model PE3590) were placed over the distal and proximal muscle belly of the VL. Electrode position was registered as the distance from the proximal border of the patella and used during all test bouts. Twitch contractions were evoked on the passive muscle using electrical stimulation consisting of single square-wave pulses of 0.1-ms duration delivered by a direct current stimulator (Digitimer Electronics, model DS7). Stepwise increments in current were delivered, separated by rest periods of 30 s, until no further increase in twitch amplitude was seen (33), and then three maximal twitches were obtained. The twitch force signal was sampled at 1,000 Hz. During the process of offline analysis, the signals were digitally low-pass filtered at 25 Hz, by use of a fourth-order zero phase lag Butterworth filter (62). Peak twitch rate of force development was determined as the peak slope in the rising part of the twitch curve derived in successive 2-ms intervals.

**Level of Habitual Activity**

Subjects were interviewed orally about their level of recreational activity at the time of recruitment, after the resistance training period, and again after the period of detraining.

**Statistical Analysis**

Repeated-measures ANOVA and subsequent Bonferroni post hoc analysis were used to answer the question whether selected parameters changed over time. Values are reported as means ± SE unless otherwise stated. The Ciba-Geigy coefficient of variation between testing sessions was calculated for main parameters in the control group (24).

**RESULTS**

**Training**

In response to 3 mo of resistance training, isokinetic muscle strength at 30 and 240°/s increased 18% ($P < 0.01$) and 10% ($P < 0.05$), respectively (Fig. 1). Anatomical quadriceps CSA increased 10% ($P < 0.001$) (Fig. 2). Agonist EMG at 30°/s increased 10%, whereas antagonist EMG remained unchanged (Table 1).

The proportion of MHC IIX decreased from 5.6 ± 0.8 to 0.8 ± 0.3% ($P < 0.001$), whereas that of MHC IIA increased from 34.0 ± 2.5 to 39.4 ± 2.0% ($P < 0.001$) (Fig. 3). All other parameters remained unchanged (velocity, acceleration, power

**Table 1. EMG activity in the training group**

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
<th>Post-Detr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quad EMG, µV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30°/s</td>
<td>267±20</td>
<td>293±19*</td>
<td>255±20</td>
</tr>
<tr>
<td>240°/s</td>
<td>383±40</td>
<td>368±26</td>
<td>377±33</td>
</tr>
<tr>
<td>Unloaded</td>
<td>409±58</td>
<td>555±20</td>
<td>354±27</td>
</tr>
<tr>
<td>Normalized Quad EMG, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30°/s</td>
<td>71±4</td>
<td>81±4*</td>
<td>71±4</td>
</tr>
<tr>
<td>240°/s</td>
<td>100±0</td>
<td>100±0</td>
<td>100±0</td>
</tr>
<tr>
<td>Unloaded</td>
<td>107±9</td>
<td>96±5</td>
<td>94±6</td>
</tr>
<tr>
<td>Antagonist Hams EMG, µV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30°/s</td>
<td>50±9</td>
<td>46±12</td>
<td>54±12</td>
</tr>
<tr>
<td>240°/s</td>
<td>59±8</td>
<td>49±8</td>
<td>42±5</td>
</tr>
<tr>
<td>Unloaded</td>
<td>65±14</td>
<td>62±8</td>
<td>63±10</td>
</tr>
</tbody>
</table>

Values are means ± SE; $n = 14$ subjects. Pre, pretraining; Post, postraining; Post-Detr, post-detraining; Quad, quadriceps; Hams, hamstring. Electromyograph (EMG) activity increased only at the slow contraction after resistance training and decreased to training levels after detraining. Antagonist hamstring EMG activity remained unchanged. *Increase from pre-to postraining ($P < 0.05$).
and EMG during maximal unloaded knee extension, evoked muscle twitch properties, weight of the lower leg).

**Detraining**

After 3 mo of detraining muscle CSA (Fig. 2), isokinetic muscle strength and power at 30 and 240°/s (Fig. 1, Fig. 4) and agonist EMG at 30°/s (Table 1) decreased to pretraining levels. Angular velocity, acceleration, total moment of force, and power in the 60–80° knee joint interval during maximal unloaded knee extension increased 10% (P < 0.05, Fig. 1), 34% (P < 0.05, Fig. 5), 31% (P = 0.01, Fig. 1), and 44% (P < 0.05, Fig. 4), respectively.

Peak velocity of maximal unloaded knee extension increased 14% (P < 0.05, Fig. 6), and occurred at a more extended knee joint angle after detraining (55 ± 2 vs. 51 ± 1°, P < 0.05). The time from onset of movement to peak velocity was unchanged from pretraining to posttraining to post-detraining (88 ± 2, 86 ± 3, and 86 ± 2 ms, respectively).

Electrically evoked peak twitch rate of force development increased 23% (P < 0.001, Fig. 7). MHC IIX increased from 0.8 ± 0.3 to 7.7 ± 1.1%, which was significantly higher compared with both pre- (P < 0.05) and postraining levels (P < 0.001) (Fig. 3). The overall proportion of MHC II (IIA + IIX) increased from 40.2 ± 2.1% to 44.7 ± 2.2% (P = 0.05) (Fig. 3). Habitual physical activity during the period of detraining remained unchanged compared with that before the training intervention and did not vary from the end of the training period until the end of the follow-up detraining period.

**Control Group**

In the nontrained control group, all parameters remained unchanged throughout the study period (see Fig. 6 for peak velocity during unloaded knee extension, Fig. 7 for twitch characteristics, and Table 2 for EMG). In the 60–80° range of motion, moment of force at 30°/s was 236 ± 13, 232 ± 13, and 219 ± 13 Nm, respectively. **Fig. 3.** Myosin heavy chain (MHC) isoform composition. After 3 mo of resistance training, the overall proportion of MHC II (IIA + IIX) remained unchanged; however, a subtype transition from MHC IIX to IIA occurred. *P < 0.001. **After 3 mo of detraining, MHC IIX increased to levels that were higher compared with pre- (P < 0.05) and postraining (P < 0.001). *Furthermore, the overall proportion of MHC II (i.e., IIA + IIX) was increased at postdetraining (P = 0.05).

**Fig. 4.** In vivo power-velocity relationship (average power and velocity in the 60–80° range of motion). Power was increased at slow-to-medium velocities after resistance training. After 3 mo of detraining, power decreased during slow-to-medium velocities but was markedly increased during very high velocity, i.e., maximal unloaded contraction. Values are group means ± SE. *Postraining > pretraining and postdetraining, P < 0.05. †Postdetraining > pre- and postraining, P < 0.05.

**Fig. 5.** Angular acceleration during maximal unloaded knee extension (average values in the 60–80° range of motion). After 3 mo of detraining, angular acceleration was markedly increased during unloaded knee extension in the training group (TG) and unchanged in the control group (CG). Values are group means ± SE. *Postdetraining > pre- and postraining, P < 0.05.
224 ± 13 N·m at pretraining, posttraining, and post-detraining, respectively. Moment of force at 240°/s was 145 ± 6, 148 ± 3, and 141 ± 7 N·m. Coefficient of variation of selected parameters ranged from 3.3 to 11.6% between testing sessions (Table 3).

**DISCUSSION**

The main and novel findings of the present study were that, after 3 mo of detraining, maximal unloaded knee extension velocity and power increased. This was seen along with faster muscle twitch contractile properties and increased proportion of fast muscle MHC.

**Effects of Resistance Training**

It is well known that resistance training induces muscle hypertrophy (3, 29, 49), gains in isokinetic muscle strength (5, 7, 19, 49), and increased agonist EMG activity (5, 13, 46). The results of the present study are in accordance with these findings. The most pronounced strength gains occurred at the slow contraction velocity (30°/s), whereas the increase was less pronounced at medium velocity (240°/s) and performance was unchanged during maximal unloaded contraction (velocity = 700–900°/s). Furthermore, EMG increased only at the slow velocity. The velocity of the resistance training exercises ranged between 30 and 90°/s, i.e., lifting the weight stack in 1–3 s through a 90° range of motion. Thus the present results support the concept of velocity-specific neural training adaptations (22, 38, 45, 50), although this concept has not been supported by all previous studies (6, 12, 44). The present results could also reflect a training-induced neural disinhibition of inhibitory mechanisms that normally may limit maximal muscle force exertion in untrained subjects (5). Thus, whereas muscle hypertrophy probably enhanced muscle strength at slow and medium contraction velocities, the present results indicate that neural adaptations further enhanced slow muscle strength. The potential nature of the neural adaptation mecha-

---

**Table 2. EMG activity in the control group**

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
<th>Post-Detr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quad EMG, µV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30°/s</td>
<td>223±25</td>
<td>235±40</td>
<td>254±44</td>
</tr>
<tr>
<td>240°/s</td>
<td>390±42</td>
<td>391±57</td>
<td>408±52</td>
</tr>
<tr>
<td>Unloaded</td>
<td>414±61</td>
<td>412±69</td>
<td>445±96</td>
</tr>
<tr>
<td>Normalized Quad EMG, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30°/s</td>
<td>62±5</td>
<td>61±4</td>
<td>64±5</td>
</tr>
<tr>
<td>240°/s</td>
<td>100±0</td>
<td>100±0</td>
<td>100±0</td>
</tr>
<tr>
<td>Unloaded</td>
<td>106±6</td>
<td>105±6</td>
<td>109±8</td>
</tr>
<tr>
<td>Antagonist Hams EMG, µV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30°/s</td>
<td>49±5</td>
<td>45±4</td>
<td>46±4</td>
</tr>
<tr>
<td>240°/s</td>
<td>62±7</td>
<td>70±14</td>
<td>60±8</td>
</tr>
<tr>
<td>Unloaded</td>
<td>50±13</td>
<td>47±12</td>
<td>58±17</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 10 subjects. No change occurred in the control group.

**Table 3. Coefficient of variation of major parameters from pre- to posttraining and from posttraining to post-detraining in the nontrained control group**

<table>
<thead>
<tr>
<th></th>
<th>Unloaded Knee Extension, %</th>
<th>Isokinetic, %</th>
<th>Twitch tRFD, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>1–2</td>
<td>2–3</td>
<td></td>
</tr>
<tr>
<td>α</td>
<td>3.6</td>
<td>4.1</td>
<td>5.4</td>
</tr>
<tr>
<td>ω</td>
<td>7.4</td>
<td>10.6</td>
<td>3.3</td>
</tr>
<tr>
<td>MT</td>
<td>6.7</td>
<td>9.1</td>
<td>9.8</td>
</tr>
<tr>
<td>Power</td>
<td>11.6</td>
<td>11.6</td>
<td>5.5</td>
</tr>
<tr>
<td>30°/s</td>
<td>5.4</td>
<td>5.5</td>
<td>3.6</td>
</tr>
<tr>
<td>240°/s</td>
<td>3.3</td>
<td>3.6</td>
<td>10.9</td>
</tr>
</tbody>
</table>

CV, coefficient of variation; 1–2, pre- to posttraining; 2–3, posttraining to post-detraining; n = 10 subjects, α, Knee extension angular velocity; ω, rotational acceleration; MT, total moment of force; tRFD, twitch rate of force development.
nisms has been discussed in detail elsewhere (1, 13, 25, 40). Finally, it should be noted that measurements of surface EMG should be interpreted with caution (26). Increased EMG amplitude in response to training is commonly believed to reflect increased recruitment and firing rate of motor units (56), which in turn would increase force output of the muscle. However, increased EMG could also reflect increased synchronization of motor units (23, 63), which may not, per se, increase muscle force. In addition, a training-induced change in muscle architecture via an increased muscle fiber pennation angle could have contributed to the present strength gains (3, 39).

Performance of maximal unloaded knee extension remained unchanged in response to the present resistance training protocol that involved 6–12 RM loads and a total of 12–15 sets for the quadriceps muscle per training session, which was designed to induce muscle hypertrophy (57). Although the training-induced increase in muscle strength and size per se may have augmented contractile impulse and thus velocity during unloaded knee extension, this benefit may have been counteracted by the almost complete removal of MHC IIX (Fig. 3). Perhaps other types of training, e.g., power training (30, 32, 40), intended ballistic contractions (12), and resistance training involving very heavy loads (1–8 RM) (6, 51, 52) may be effective to improve muscle force generation at very high contraction velocities through increased neural drive and/or selective hypertrophy of type II muscle fibers.

Effects of Detraining

After 3 mo of detraining, the training-induced strength gains at 30 and 240°/s returned to pretraining levels along with muscular atrophy (Fig. 2) and decreased EMG at the slow velocity (Table 1). These results are in line with previous studies (8, 29, 35, 49). However, most importantly, maximal unloaded movement speed and power increased after the period of detraining (Fig. 1, Fig. 4) concurrently with an increase in the proportion of fast MHC (Fig. 3).

Agnostic and antagonist EMG activity during the maximal unloaded limb movement remained unchanged throughout the study, indicating that peripheral adaptations alone were responsible for the observed increase in maximal unloaded limb movement speed. A higher electrically evoked twitch rate of force development was seen with detraining (Fig. 7), which suggests that muscle excitation-contraction coupling and/or cross-bridge cycling rate was enhanced. A similar finding has been reported for human calf muscle after 8 wk of detraining (37). Causative factors could be related to altered sarcoplasmic reticulum Ca$^{2+}$ kinetics (18, 42) and MHC isoform composition (33). In line with previous findings (8), detraining increased the levels of MHC IIX compared with those seen before resistance training. Furthermore, the present study suggests that the overall proportion of fast MHC II (IIA + IIX) may increase at the expense of decreased MHC I content with detraining. Such a MHC transition has also been observed in healthy subjects exposed to bed rest (9). However, in the present study, the subjects resumed their previous activity level during the period of detraining. Together this indicates that a marked decrease in the level of activity induces a shift toward a faster MHC isoform expression (I → IIA → IIX). It has been proposed that human skeletal muscle MHC IIX represents a default gene setting that is expressed in the absence of muscle contractile activity (9, 11, 28). The present results extend this hypothesis and suggest that the relative decrease in activity triggers this response. In support of this and in line with previous findings (8), withdrawal of resistance training stimulus for 3 mo triggered a shift toward faster MHC phenotypes (I → IIA → IIX) (Fig. 3).

Physical training as well as decreased activity per se do not appear to affect contractile characteristics of individual MHC isoforms (61), although individual muscle fibers can change the MHC isoform expressed (8, 10, 28). Thus, regardless of the level of conditioning, type IIX muscle fibers contract approximately twice as fast as type IIA and 9- to 10-fold faster than type I muscle fibers, whereas hybrid fibers display intermediate contractile properties (16, 17, 43). Likewise, intermediate contractile properties of whole muscle in vivo with a mixed muscle fiber-type composition are observed compared with either a type I or type II dominated muscle (33). At firsthand, the change in MHC isoform composition from the trained state to the detrained state appears small (Fig. 3) compared with the change in unloaded limb movement speed (Figs. 1 and 6). However, a rough estimate based on a 1:4:9 relationship in maximal contraction velocity between MHC type I-IIA-IIX (16, 17, 43) indicates that the present change in MHC composition from posttraining to post-detraining could theoretically induce a relative increase of 21% in maximal unloaded contraction velocity, which corresponds well to the observed 14%:

\[
\frac{(%\text{MHC I} \times 1)_{\text{detrained}} + (%\text{MHC IIA} \times 4)_{\text{detrained}} + (%\text{MHC IIX} \times 9)_{\text{detrained}}}{(%\text{MHC I} \times 1)_{\text{trained}} + (%\text{MHC IIA} \times 4)_{\text{trained}} + (%\text{MHC IIX} \times 9)_{\text{trained}}} = \frac{(55.3\% \times 1) + (37.1\% \times 4) + (7.7\% \times 9)}{(59.8\% \times 1) + (39.4\% \times 4) + (0.8\% \times 9)} = 1.21
\]

It should be noted that this calculation is based on maximal contraction velocities of single fibers (16, 17, 43) and should therefore be taken with caution, because function of whole muscles in vivo is more complex than that of single fibers in vitro. Nevertheless, a phenotypic transition toward faster MHC isoforms in response to detraining could theoretically explain the present finding of faster unloaded limb movement speed. In line with the present findings, it has previously been found that muscle fiber-type composition becomes increasingly more related to contractile performance as the velocity of movement increases (2, 21, 58), although not clearly supported by all studies (36). Tihanyi et al. (60) demonstrated that, when velocity of shortening exceeds half of maximal, fiber-type-related differences may become discernible for contractile quadriceps strength in vivo. Likewise, Thorstensson et al. (58) found a significant correlation between the percentage of type II muscle fibers and maximal knee extension velocity. The present results support that adaptations of the muscle at the molecular level play a prominent role during high-velocity movements with negligible external resistance. Thus it is likely
that increased velocity and power of maximal unloaded limb movement occurred mainly as a result of the observed MHC transition toward faster isoforms. It cannot be ruled out, however, that other factors could also have contributed to the present findings, e.g., enhanced SR Ca\(^{2+}\) kinetics or excitation-contraction coupling.

Muscle power output is an important functional parameter as it is the product of muscle force and contraction velocity (i.e., \(P = M_T \times \omega\)). Power during unloaded knee extension was markedly increased after detraining (Fig. 4). Increased rotational acceleration of the lower leg (Fig. 5) affects both velocity (i.e., \(\alpha = \omega \times dr\)) and moment of force (i.e., \(M_1 = \alpha \times I\)), which together affect power. During unloaded knee extension, peak velocity is achieved in less than 90 ms. In contrast, longer time is needed to reach maximum muscular force (>300 ms, e.g., Ref. 59). Thus during unloaded limb movement, a high contractile rate of force development (4) is of vital importance to achieve a high acceleration and thus power and velocity. The greater cross-bread cycling rate of type IIA and type IIX muscle fibers compared with type I muscle fibers (17) allows for a faster rate of force development and thus enhanced acceleration of the lower limb. In support of this, Houston and coworkers (36) found that angular acceleration of maximal unloaded knee extension was significantly correlated to muscle fiber-type composition. Thus the marked increase in power likely reflects an increased rate of force development, which in turn allows for a greater acceleration of the lower limb. Other studies have reported decreased level of power in response to detraining (41, 47). The discrepancy between the present and previous results may be related to the method of measuring power. In the present study, power development was measured during conditions of no external resistance. However, in the previous studies, movement of the leg was performed against external resistance. Power development against external resistance has been shown to be modestly to strongly correlated to maximal muscle strength (14, 55). Thus these previous reports of decreased power in response to detraining (41, 47) may be associated with decreased maximal muscle strength. Furthermore, it is unknown whether an overshoot of fast muscle MHC occurred in response to detraining in these previous studies. In agreement with these previous findings, power was observed to decrease in the present study at medium and slow isokinetic muscle contraction where a high moment of force was present (Fig. 4). Thus, when muscle contraction is performed against substantial external load, power production is highly influenced by muscle mass and maximal contraction strength (55). In contrast, when maximal velocity unloaded movements are performed intrinsic muscle contractile properties appear to dominate.

**Implications for Performance**

Some care should be taken when interpreting the functional relevance of the present results for athletic performance. Highly trained athletes have shown preferential atrophy of type II muscle fibers with 2 wk of detraining (34), which would reduce the relative proportion of type II MHC. Thus the present findings may not extend to highly trained athletes because of differences in initial muscle fiber size and level of conditioning. It should also be mentioned that muscles do not always function at maximal angular velocities and in unloaded fashion. Thus it can be speculated that the present results may only relate to high-velocity unloaded movements, e.g., punching, kicking, or smashing, but not to loaded movements like sprint running (load of body weight), jumping, and shot putting. Consequently, further investigations should be conducted in trained athletes involved in sports with high-velocity unloaded movements.

In conclusion, the present study demonstrated that detraining from resistance training increases velocity and power of maximal unloaded limb movement in previously untrained subjects. This was seen along with increased expression of faster muscle MHC isoforms (I \(\rightarrow\) IIA \(\rightarrow\) IIX) paralleled by faster intrinsic contractile properties.

**ACKNOWLEDGMENTS**

Thanks go to Hanne Overgaard for technical assistance.

**GRANTS**

This study was supported by the Danish Elite Sport Association (Team Danmark).

**REFERENCES**


